Clinical Profile Of Acute Myocardial Infarction Patients At PNS -SHIFA Hospital Karachi

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ABSTRACT:

Objective: To study the risk factors, complications and use of streptokinase in patients of acute myocardial infarction (MI) presenting at Coronary Care Unit (CCU) of PNS SHIFA Hospital Karachi.

Materials and Methods: This study was conducted at CCU, PNS SHIFA Hospital Karachi, from January 2008 to December 2012. It is a retrospective cohort study with non-probability convenient sampling. Diagnosed MI cases were included in the study from all age groups, gender and backgrounds. Patients with other cardiac/non-cardiac diseases presenting with symptoms similar to MI were excluded.

Results: Acute myocardial infarction patients were 418, out of the total 2250 admissions in CCU, 71.29% were males. 67.7% were smokers, 60.2% had sedentary life style, 56.2% patients were known hypertensive and 42.1% were known diabetics. 63.63% were eligible for streptokinase administration at the time of admission. Left ventricular failure developed in 13% patients and 6.6% went into cardiogenic shock.

Conclusion: Acute myocardial infarction was found to be fairly common cardiac emergency among all cases admitted in the CCU. It has strong association with male gender, smoking, sedentary life style, hypertension, diabetes mellitus and hyperlipidemia. Left Ventricular failure was the leading complication. 63.63% were eligible for streptokinase administration and mortality rate was 5%.

Keywords: Acute myocardial infarction, Streptokinase, Clinical profile, Risk factors, Complications.

INTRODUCTION:

Acute Myocardial Infarction (AMI) is a clinical syndrome resulting from a coronary artery occlusion with cardiac myocyte death in the region supplied by the culprit artery[1]. WHO has estimated in 2004 that 12.2% (7.2 million) of worldwide deaths were from ischemic heart disease with it being the leading cause of death in high or middle income countries and second only to lower respiratory infections in lower income countries.[2] Cardio-vascular diseases are a significant health burden on a country or as general on the health system. In the year 2005, 58 million people died of cardiovascular diseases (CVD) accounting for 30% of all deaths worldwide; more than half of these deaths were in the developing countries[3]. Characteristic presentation of acute myocardial infarction (AMI) is with central chest pain radiating to neck, jaw and upper or even lower arm. The pain is typically dull constricting, choking or heavy and may also be associated with breathlessness, sweating and nausea[4]. The WHO criteria were refined in 2000 to give more prominence to cardiac biomarkers. According to new guidelines cardiac troponin rise accompanied by either typical symptoms, pathological Q waves, ST elevation or depression, or coronary intervention is diagnostic of MI[5]. Over the years, careful monitoring of the Framingham Study population has led to the identification of major CVD risk factors, as well as valuable information on the effects of these factors such as hypertension, [6] obesity, smoking[7] age, gender, and psychosocial issues. Diabetes[8] and lack of physical activity [9] are also important risk factors. Coronary artery occlusion is considered the basis for and consequence of myocardial ischemia. In most cases of acute MI, permanent damage to the heart occurs when the perfusion of the myocardium is severely reduced for an extended interval of (usually lasting 2 to 4 hours). This delay in the onset of permanent myocardial injury provides the rationale for rapid diagnosis in acute MI to permit early coronary reperfusion, the purpose of which is to establish reperfusion and salvage as much at risk myocardium as possible.[10]

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MATERIALS & METHODS:

The study was conducted at PNS-SHIFA hospital Karachi, a tertiary care hospital from January 2008 to December 2012. It is a retrospective cohort study in which 418 patients were included presenting at the coronary care unit of PNS SHIFA with acute myocardial infarction. The sampling technique was non-probability convenient sampling. All the cases in which diagnosis of MI was confirmed by clinical evaluation and laboratory studies were included in the study. Patients having other cardiac or non-cardiac diseases presenting with symptoms similar to MI were not included.

The patients were managed according to Standard PNS-SHIFA protocols (AHA/ACCA guidelines) for emergency management of AMI. The diagnosis was made on the basis of targeted history, examination, ECG changes and elevated cardiac enzymes/markers (CK-MB, Troponin). The ECG changes included ST segment elevation over the area of damage, ST segment depression in leads opposite to infarct, pathological Q waves, reduced R waves and inverted T waves. Cardiac enzyme CK MB was found out in Units per Liter by using International Federation of Clinical Chemistry (IFCC) method using P 800 (cobas) by company Roche, Trop I was done using Electro Chemiluminescence immunoassay (Eclia) using Cobas E411 by Roche. Treatment was started without any delay after establishing the diagnosis. Anti-platelet agents Aspirin 160-325 mg orally and Clopidogrel 600 mg orally were given.[10] In hypoxemic patients oxygen 2-4 liters per minute was given by nasal prongs or face mask and the need for oxygen administration was reassessed after 6-8 hours. Sublingual Nitroglycerine

was given in a dose of 0.4 mg up to three doses 5 minutes apart. Oral Beta Blocker Metoprolol 25-50 mg 6 hourly was given. Streptokinase was administered 1.5 million units per 1 hour infusion to patients presenting within first 10-12 hours of AMI and met the streptokinase eligibility criteria.[11] Angiotensin converting Enzyme Inhibitors Captopril was also given.[12] After stabilizing the patients, a detailed history was obtained regarding predisposing risk factors. The anatomical location of infarct was determined on the basis of 12 lead ECG findings. The patients were observed for development of any complications and managed accordingly.[13] The data was collected on a predesigned proforma after obtaining an oral consent from the patients. Data was analyzed using SPSS 15 software and results are expressed as percentages, frequencies.

RESULTS:

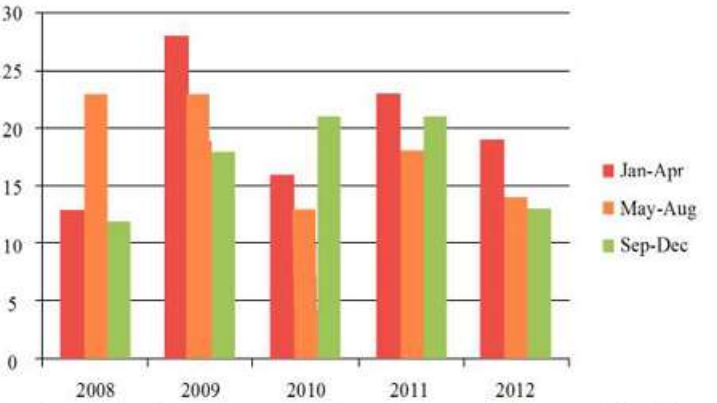
A total of 2250 patients were admitted at CCU of PNS SHIFA, out of which 1076 (47.8%) had Acute Coronary Syndrome and 418 suffered from Acute Myocardial Infarction which makes 18.5% of total patients and 38.8% of ACS patients. As shown in table 1, out of 418 AMI patients 298 were males and 120 were females. 157 out of 418 were between the ages of 46 to 60 years (Table 1)

Table: 1
Non-Variable and Variable Risk Factors
N=418

Non-Variable:				
Gender	Males	Females		
	71.29%	28.70%		
Age(years)	30-45	46-60	61-75	76-90
	16.2%	37.5%	33.2%	13.1%
Genetic predisposition (family history)	31.8%			
Variable:				
Smokers	67.7%			
Sedentary life style	60.2%			
Hypertension	56.2%			
Diabetes	42.1%			
Hyperlipidemia	37.8%			
Psychosocial Factors	20.3%			

Out of 418 AMI patients 266(63.63%) met the eligibility criteria for streptokinase administration while 152(36.36%) patients were not given streptokinase as they did not meet the criteria or presented late at the CCU. 276(66%) patients suffered from Anterior Wall MI, 121 (28.9%) had Inferior Wall MI and only 21 (5%) suffered from lateral wall MI. (Figure 1)

Figure: 1
Quarterly Strength of Streptokinase Eligible AMI patients



Left ventricular failure was the most common complication which was seen in 56 out of 418 (13%) followed by complete heart block, 38 patients out of 418 (9%), cardiogenic shock occurred in 28 patients (6.6%) and death occurred in 21 patients (5%). 275 out of 418 had an uneventful recovery to be discharged from CCU with follow up visits at OPD for long term management or referral to Armed Forces Institute of Cardiology(AFIC) Rawalpindi or National institute of cardiovascular diseases (NICVD) Karachi for further evaluation and coronary interventions (Table 2)

Table: 2
Complications of Acute Myocardial Infarction

Complications	Percentage (%)
Left Ventricular Failure	13
Complete Heart Block	9
Cardiogenic Shock	6.6
Death	5

DISCUSSION:

An Acute Myocardial Infarction (AMI) requires immediate medical attention. Treatment attempts to salvage as much myocardium as possible and to prevent further complications, hence the phrase "time is muscle".[12] Antiplatelet regimen is started as soon as diagnosis of AMI is established. It includes Aspirin 160-325 mg loading dose in acute setting followed by 75 mg daily, continued indefinitely have proven to be beneficial in decreasing mortality. It acts by irreversible acetylation of cyclooxygenase which inhibits synthesis of thromboxane A^2 leading to rapid decrease in its plasma levels thus interfering with platelet aggregation.[14,15] Clopidogrel 600mg has been associated with reduction in infarction size compared with lower dose of 300 mg and it should be continued for up to 1 year after infarction at dose of 75mg per day. It acts by irreversibly blocking ADP receptor(P2Y12) on platelet.[15,16] There is less data to support or refute use of oxygen therapy in the care of AMI patients, its use is particularly restricted to hypoxemic patients (oxygen saturation less than 90%), according to a pooled Cochrane analysis of 3 trials there is 3 fold higher risk of death of patients with AMI treated with oxygen than patients treated at room

temperature.[11,12] Nitroglycerine causes smooth muscle relaxation, thus decreasing myocardial oxygen demand by reducing pre-load and increasing myocardial oxygen supply by dilatation of infarcted and collateral coronary vessels. It also relieves or diminishes chest discomfort.[17] Fibrinolysis is the most effective pharmacological means to achieve artery patency leading to coronary reperfusion limiting infarct size, decreasing left ventricular dysfunction and improving survival.[20,21] Fibrinolytic therapy ideally should me administrated within first 30 minutes (door to needle time is 30 minutes) or up to 12 hours of myocardial infarction to patients who meet the eligibility criteria. Fibrinolytic agents work by promoting conversion of plasminogen to active plasmin form which lysis the fibrin clot. Absolute contraindications to fibrinolytic therapy include known structural cerebral vascular lesion, malignant intracranial neoplasm, ischemic stroke within 3 months except acute ischemic stroke within 4.5 hours, significant closed-head or facial trauma within 3 months, suspected aortic dissection. Active bleeding or bleeding diathesis, intracranial or spinal surgery within 2 months, severe uncontrolled hypertension, prior streptokinase treatment in last 6 months. In our study 63.63% met the eligibility criteria and were timely administered streptokinase, while 36.36% patients did not meet the criteria or presented late at CCU. Late presentation was the main reason for ineligibility for streptokinase, because of lack of awareness and education and people presenting from rural areas where these facilities are not available.[22] Aspirin and Clopidogrel should be administered to patients with AMI who receive fibrinolytic therapy for better outcome level of evidence is according to American Heart Association guidelines for treatment of ST Elevation Myocardial Infarction.[12] Intravenous Beta Blockers should be started in first 24 hours of AMI who are not at risk of cardiogenic shock, have signs of heart failure or evidence of low output state, they help reduce pain and reduce risk of re-infarction or ventricular fibrillation.[12,18] Angiotensin Converting Enzyme (ACE) Inhibitors leads to reduction in ventricular remodeling and dilatation with subsequent decrease in congestive heart failure. Angiotensin converting enzyme inhibitors in the convalescent phase more than three days after the onset of symptoms) has also been well documented in clinical trials. [19,20,21,22] The administration of these drugs during the healing phase of infarction and thereafter in patients with left ventricular ejection < 40% whether symptomatic or not reduces morbidity and mortality.[23] ACE inhibitors are now recommended in every patient with acute MI, to be started within first 24 hours and continued up to six weeks, unless there are contraindications[24]

Our most common complication was Ventricular failure that developed in 13% of patients soon after presentation that emphasizes on the role of ACE inhibitors in the emergency management, followed by complete heart block in 9% of patients, cardiogenic shock went on developing in 6.6% of the patients. Despite all our efforts and treatment, 5% of patients did not survive because of extensive damage to the myocardium, late presentation or development of complications. One of the studies has documented a mortality rate of 10.5% [20]

Our study showed that males are at more risk than women and the highest incidence of AMI presentation was between ages of 46 to 60. Yusuf et al has concluded median age of AMI in South Asia to be 53 (46-61) which is close to our findings. [25] Among the variable risk factors in our study, smoking has the highest association 67.7% followed by sedentary life style 60.2% then was hypertension 56.2%, diabetes 42.1% and 37.8% hyperlipidemia. Pais et al has also related smoking as the strongest variable risk factor followed by diabetes and hypertension which is similar to our findings.[26] Rosengren et al in their study has associated psychosocial stressors with AMI and our study showed 20.3% patients had psychosocial factors affecting their lives highlighting the importance of addressing these stressors in the preventive strategies. [27,28]

CONCLUSION:

Myocardial infarction (MI) is a fairly common cardiac emergency of all cases admitted in PNS SHIFA CCU. It has strong association with male gender, smoking sedentary life style, hypertension, diabetes mellitus and hyperlipidemia. Left Ventricular failure was the leading complication. 63.63% were found to be eligible for streptokinase administration and mortality rate was 5%.It is important to identify high risk groups, keep them under medical surveillance and if presentation is with AMI the variable risk factors should tried to be modified.

REFERENCES:

1. Antman EM: ST-segment elevation myocardial infarction Pathology, Pathophysiology, and clinical Features In: Bonow RO, Mann DL, Zipes DP, Libby P, eds. Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine, 9thed, Philadelphia, PA:Saunders Elsevier; 2011:chap 54.
2. World Health Organization The Global Burden of Disease: 2004 Update. Geneva: World Health Organization 2008. ISBN 92-4-156371-0
3. Antman EM, Anbe DT, Armstrong PW, Bates ER, Green LA, Hand M et al. ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction. J Am Coll Cardiol 2004;44 (3): 671-719 doi:10.1016/j.jacc.2004.07.002.PMID 15358045
4. Newby D.E., Grub N.R., Bradbury A. Cardiovascular Disease.In:. Nicki R. Colledge, Brian R. Walker , Stuart H. Ralston, editors. Davidson`s Principles and Practice of Medicine Churchill livingstone An imprint of Elsevier. 21st edition 2010. p 535-6
5. Myocardial infarction redefined—a consensus document of The Joint European Society of Cardiology/American College of Cardiology Committee for the redefinition of myocardial infarction. J Am Coll Cardiol 2000;36 (3):959-69
6. Antman E M, Slewyn AP, Braunwald E, Loscalzo J. Ischemic Heart Disease In: Fauci, Braunwald, Kasper, Hauser, Longo, Jameson, Localzo, editors. Harrison's Principles of Internal Medicine, 17th edition p 1514-25

7. Buse JB, Ginsberg HN, Bakris GL. Primary prevention of cardiovascular diseases in people with diabetes mellitus: a scientific statement from the American Heart Association and the American Diabetes Association. Circulation115 (1):114-26 doi:10.1161/CIRCULATIONAHA.106.179294. PMID 17192512
8. Graham I, Atar D, Borch-Johnsen K. European guidelines on cardiovascular disease prevention in clinical practice: executive summary: Fourth Joint Task Force of the European Society of Cardiology and Other Societies on Cardiovascular Disease Prevention in Clinical Practice (Constituted by representatives of nine societies and by invitedexperts). Eur.HeartJ.2007; 28 (19):2375–414 doi:10.1093/eurheartj/ehm316. PMID 17726041
9. Schoen FJ, Mitchell RN. The Heart. In: Kumar V, Abbas AK, Fausto N, Aster JC, editors. Robbins and Cotran. 8th ed. South Asia: Saunders Company; 2010. pp 529-49.
10. Siddiqui A H, Kayani A M. Acute myocardial infarction-clinical profile of 1000 cases. Pakistan heart Journal. 2000;32&33 (1,2 & 4): 42-5
11. O'Connor RE, Brady W, Brooks SC. "Part 10: acute coronary syndromes: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care". Circulation 2010;122 (18 Suppl 3): S787–817. doi:10.1161/CIRCULATIONAHA.110.971028. PMID 20956226
12. O'Gara P T, Kushner FG, Ascheim DD, Casey DE, Chung MK, de Lemos JA, et al. ACCF/AHA Guideline for the management of ST- Elevation Myocardial Infarction. Copyright 2013, American College of Cardiology Foundation and the American Heart Association, Inc. J Am Coll Cardiol. 2013;61(4):e78-e140.Doi: 10.1016/j.jacc.2012.11.01
13. Collaborative meta-analysis of randomized trials of antiplatelet therapy for prevention of death, myocardial infarction, and stroke in high risk patients. BMJ. 2002;324:71-86
14. Zhender J L. Drugs used in Disorders of Coagulation In: Betram G. Katzung, Susan B. Masters, Anthony J. Trevor, editors Basic and Clinical Pharmacology, 11th Edition. Lange TATA McGRAW-HILL EDITION. P 598-9
15. Patti G., Bárczi G., Orlic D. Outcome comparison of 600- and 300-mg loading doses of clopidogrel in patients undergoing primary per-cutaneous coronary intervention for ST-segment elevation myocardial infarction: results from the ARMYDA-6 M. J Am Coll Cardiol. 2011;58:1592-9
16. Chen Z.M., Pan H.C., Chen Y.P., Early intravenous then oral metoprolol in 45,852 patients with acute myocardial infarction: randomized placebo-controlled trial. Lancet. 2005;366:1622-32
17. ISIS Collaborative group (1993) ISIS IV. Randomized study of oral captopril in over 50,000 patients with suspected, acute myocardial infarction. Circulation, 88, 1-394.
18. The CONSENSUS Trial Study Group effects of enalapril on mortality in severe congestive heart failure, results of the Cooperative North Scandenavian. Enalapril survival study (CONSENSUS) N. Eng. Journal of Med. 1987;316, 1429-35
19. N Sharpe, Murphy J, H Smith , S Hannan . Treatment of patients with symptomless left ventricular dysfunction. Lancet. 1988; 256-9.
20. Gruppo Italiano per to studio delta sopravvivenza nell infarctio miocardico GISSI-3 Effects of lisinopril & transdermal glyceryl trinitrate singly or together on 6-week mortality, & ventricular function after acute myocardial infarction Lancet. 1994; 343,1115-22
21. Pfeffer M.A., Braunwald E., Moyé L.A., SAVE Investigators Effect of captopril on mortality and morbidity in patients with left ventricular dysfunction after myocardial infarction: results of the Survival and Ventricular Enlargement Trial. N Engl J Med. 1992;327:669-77
22. Armstrong PW, Colleen D. Fibrinolysis for Acute Myocardial Infarction Current Status and New Horizons for Pharmacological Reperfusion, Part 1. American Heart Association. Circulation 2001; 103: 2862-6 doi: 10.1161/01.CIR.103.23.286
23. Braunwald E, Cannon CP. The open-artery theory is alive and well—again. N Engl J Med.1993; 329:1650-52.
24. Antman EA., General hospital management in Jullan R, Braunwald E., (eds Management of cute myocardial infarction. W, B. Saunders Company 1994
25. Yusuf S, Hawken S, Ôunpuu S, Dans T, Avezum A, Lanas F, et al. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries. The Lancet, 2004; 364 (9438):937-52. Copyright © 2004 Elsevier Ltd. doi:10.1016/S0140-6736(04)17018-9
26. Pais P, Pogue J , Gerstein H , Zachariah E , Savitha D, Jayprakash SDM, et al. Risk factors for actue myocardial infarction in Indians. The Lancet.1996; 348(9024):358 - 63. doi:10.1016/S0140-6736(96)02507-X
27. Rosengren A, Hawken S, Ounpuu S, Sliwa K, Zubaid M, Almahmeed WA, et al. Association of psychosocial risk factors with risk of acute myocardial infarction. The Lancet, 2004;364 (9438): 953–62
28. Busko M. High-risk plaque predicts ACS in ER patients with chest pain. Heartwire [serial online]. July 18, 2014 http://www.medscape.com/viewarticle/828520